Rapp. P.-v. Réun. Cons. int. Explor. Mer, 169: 327-337. 1975.

DISEASES OF PINNIPEDS

S. H. Ridgway
Ocean Sciences Department, Naval Undersea Center, San Diego, California, U.S.A.
J. R. Geraci
Wildlife Disease Section, Department of Pathology, University of Guelph, Guelph, Ontario, Canada
and
W. Medway
School of Veterinary Medicine, University of Pennsylvania, Philadelphia, Pennsylvania, U.S.A.

INTRODUCTION

One of the major problems in our understanding of pinniped disease is the small number of cases actually examined. Captive pinnipeds are maintained in small groups, and even though an individual may be associated with them over many years, his total experience relative to their diseases is usually limited and often over-shadowed by husbandry and maintenance considerations. Most wild populations are found in remote areas and no one, to our knowledge, has set out specifically to make a systematic and comprehensive study of their diseases. Thus, what we have learned so far has been from incidental and often accidental findings.

This presentation deals with an overview of some of the representative diseases in pinnipeds. It is a modest and necessarily incomplete gathering from personal experience and literature sources. Ridgway (1972) has compiled a recent, more complete tabulation of pinniped diseases which the reader is invited to consult for references not included here.

CLINICAL LABORATORY TESTS

Because the signs of disease are as yet only poorly recognized in pinnipeds, clinical laboratory data have been of primary importance in diagnosis. Tables 80 and 81 give normal values for white blood cell and red blood cell measures in several pinnipeds and Table 82 gives values for various constituents in milk.

A REVIEW OF DISEASES FOUND

Ocular disease

Almost every institution that maintains pinnipeds has encountered some form of ocular disease. Eye conditions are frequently seen in wild animals as well.

The primary eye conditions which have been observed are conjunctivitus, cataracts, corneal ulceration, corneal edema and scarring.

Conjunctivitis

Conjunctivitis often occurs either as a separate entity, or in conjunction with the other eye or systemic diseases. It nearly always accompanies corneal ulceration, and has been induced spontaneously and acutely by hyperchlorination of pool water. Sometimes conjunctivitis is a sequel to a simple scratch which occurs during playful or competitive behaviour.

Whatever the inciting cause, it can lead to bacterial infection which in turn, as suggested by Gallacher and Waters (1964), may result in pneumonia due to pus inspiration.

Simple, uncomplicated conjunctivitis responds favourably to topical application and intrapalpebral injections of corticosteroids, together with local or systemic antibiotic support.

Cataracts

Cataracts have been observed in captive pinnipeds. Hubbard and Poulter (1968) proposed that Steller sea lion pups fed on a formula containing whipping cream developed bilateral cataracts, and went on to suggest that these were caused by natural sugar present in the whipping cream. It is possible that milk sugar could produce cataracts in sea lions, less likely in true seals and unlikely in young walruses, as the latter have been maintained successfully on diets rich in whipping cream, with no observable consequences (see Ridgway, 1972 for discussion of glucose metabolism in marine mammals).

Prolonged exposure to bright light, injury and parasites are other possible causes of cataracts in pinnipeds.

Table 80. Comparison of white blood cells of various pinnipeds

	N	Total WBC	Seg(%)	Non Seg(%)	L(%)	M(%)	E(%)
California sea lion[1] (*Zalophus californianus*)	8	9 232 ± 1 533	58 ± 7	6 ± 6	28 ± 10	4 ± 2	4 ± 4
Harbour seal (*Phoca vitulina*)	5	8 016 ± 1 000	59 ± 20	1 ± 1	33 ± 22	4 ± 2	1 ± 1
Grey seal[2] (*Halichoerus grypus*)	6	10 663 ± 3 063	65 ± 8		19 ± 8		2·8 ± 2·7
Harp seal[3] (*Pagophilus groenlandicus*)	13	7 821 ± 1 671					
Elephant seal[4] (*Mirounga angustirostris*)	3	9 526 ± 1 856	55 ± 11	0	38 ± 14	3 ± 1	4 ± 1

[1] S. H. Ridgway, Ed. 1972. Mammals of the sea. Biology and medicine. Charles C. Thomas, Springfield, Ill. p. 654.
[2] Geraci, J. R. 1971. Functional hematology of the harp seal (*Pagophilus groenlandicus*). Physiol. Zool., 44(3):162–70.
[3] Greenwood, A. G., S. H. Ridgway & R. J. Harrison. 1971. Blood values in young gray seals. Am. Vet. Med. Assoc., J., 159(5):571–74.

Table 81. Comparison of red blood cells in pinnipeds

	N	Diam. RBC microns	RBC $\times 10^6$/mm^3	PCV(%)	Hb(g/dl)
California sea lion[5] (*Zalophus californianus*)	8	7·1 ± 0·2	4·38 ± 0·7	45 ± 5	15·0 ± 2·1
Harbour seal[6] (*Phoca vitulina*)	5	6·4 ± 0·3	5·45 ± 0·7	52 ± 6	19·2 ± 1·3
Grey seal[1] (*Halichoerus grypus*)	6		5·04 ± 0·41	57·2 ± 5·8	19·1 ± 0·2
Harp seal[6] (adults) (*Pagophilus groenlandicus*)	15		4·57 ± 0·35	59·2 ± 3·2	25·2 ± 1·5
Crabeater seal[3] (*Lobodon carcinophagus*)	5	9	4·10 ± 0·55		18–18·5
Bladdernose (hooded) seal[7] (*Cystophora cristata*)	2		4·3 – 5·3	60–67	25·8 ± 28·4
Weddell seal[7] (*Leptonychotes weddelli*)	9		3·62– 3·85	54–65	22·5–25
Elephant seal[3] (*Mirounga angustirostris*)	13	7·6 ± 0·2	5·55 ± 0·26	55·6 ± 2·4	25·0 ± 0·0
Ribbon seal[6] (*Histriophoca fasciata*)	5		4·49	66·6	24·5

[1] Greenwood, A. G., S. H. Ridgway & R. J. Harrison. 1971. Blood values in young gray seals. Am. Vet. Med. Assoc., J., 159(5):571–74.
[2] Clausen, G. & A. Ersland. 1969. The respiratory properties of the blood of the bladdernose seal (*Cystophora cristata*). Resp. Physiol., 7: 1–6.
[3] Tyler, J. C. 1960. Erythrocytes and hemoglobin in the crabeater seal. J. Mammal., 41(4):527.
[4] Lenfant, C., K. Johansen & J. D. Torrance. 1970. Gas transport and oxygen storage capacity in some pinnipeds and the sea otter. Resp. Physiol., 9:277–86.
[5] S. H. Ridgway, Ed. 1972. Mammals of the sea. Biology and medicine. Charles C. Thomas, Springfield, Ill. p. 606.
[6] Geraci, J. R. 1971. Functional hematology of the harp seal (*Pagophilus groenlandicus*). Physiol. Zool., 44(3):162–70.
[7] Kooyman, G. L. & C. M. Drabek. 1968. Observations on milk, blood, and urine constituents of the Weddell seal. Physiol. Zool., 41(2):187–94.

Corneal ulceration

This condition is most likely to result from direct injury to the eye, such as penetration by a sharp object like the nail or tooth of another animal; it can also result from an untreated chronic conjunctivitis.

Corneal ulcerations in marine mammals are especially difficult to treat. The eye must be protected from infection and further injury. This can be accomplished by suturing the eyelids together so that medication can be applied to and held around the

Table 82. Comparison of milk composition of various pinnipeds with that of the cow

	Water	Total Solids (%)	Fat (%)	Protein (%)	Lactose (%)	Ash (%)
California sea lion (*Zalophus californianus*)	47·3	52·7	36·5	13·8	0	0·64
Northern fur seal (*Callorhinus ursinus*)	36·4	63·6	51·5	11·3	0·10	0·49
Hooded seal (*Cystophora cristata*)	49·8	50·2	40·4	6·7	–	0·86
Harp seal (*Pagophilus groenlandicus*)	45·1	54·9	46·9	6·8	0·77	0·39
Gray seal (*Halichoerus grypus*)	32·3	67·7	53·2	11·2	2·6	0·70
Weddell seal[2] (*Leptonychotes weddelli*)	43·6	56·3	42·2	–	–	–
Cow (*Bos taurus*)	87·5	12·5	4·0	3·6	4·65	0·70

[1] S. H. Ridgway, Ed. 1972. Mammals of the sea. Biology and medicine. Charles C. Thomas, Springfield, Ill. p. 687.
[2] Kooyman, G. L. & C. M. Drabek. 1968. Observations on milk, blood and urine constituents of the Weddell seal. Physiol. Zool., 41(2):187–94.

eye. When the eye has healed the sutures can be removed without ill effect. Corneal flap operations have also been performed with some degree of success. In severe injuries, or post-surgically, a small scar usually remains after the lesion heals.

Corneal edema or cloudiness

There are several possible causes for the corneal edema which has frequently been observed in pinniped colonies. Ronald, Johnson, Foster and Vander Pol (1970) state that "to prevent seals" eyes from becoming opaque, common salt (NaCl) is added to the water biweekly in a concentration of one kg per 400 litres and seals are allowed to swim in the solution for at least an hour before the tank is drained and refilled with fresh water. If any seals are found to have opaque eys, salt is immediately added to the water and the condition usually clears within a few hours."

The frequent addition of salt to seal pools is no doubt desirable, and it unquestionably does promote corneal clearing in some instances, especially in harp seals maintained in fresh water.

It must be noted, however, that many institutions keep seals and sea lions in fresh water for years without observing a great incidence of corneal opacities, and some facilities which keep animals in sea water occasionally do observe the problem. Perhaps the edema is caused by some other factor but develops more rapidly in fresh water. The relationship of corneal edema to fresh water is still open to question, at least in most species.

On separate occasions in two different facilities, sea lions and harbour seals were inadvertently exposed to abnormally high concentrations of chlorine bleach. Within an hour bilateral corneal opacities appeared in all animals, with photophobia in some. In one colony the opacities gradually disappeared over the next 48 hours during which the animals swam in sea water – with no medication. In the other, the condition remained for 2–3 weeks despite medication, or possibly even as a result of it.

A gray seal developed a severe corneal opacity in one eye and a slight opacity in the other just after surgery. The eyes were examined by an ophthalmologist who suggest that Hibitane[3], a disinfectant that was being added to the water to prevent post-surgical infection, had probably caused the opacity. The use of the disinfectant was discontinued and the eyes cleared over the next month while the seal remained in fresh water.

There are a number of other possible causes of eye conditions including toxins, nutritional imbalances, parasitic and virus diseases, all of which warrant further study.

Diseases of orphaned and stranded pinnipeds

We often encounter sick pinnipeds, stranded along the California and other sea coasts. They include sea lions, *Zalophus californianus*, young elephant seals *Mirounga angustirostris*, harbour seals *Phoca vitulina* and gray seals, *Halichoerus grypus*. The following is a tabulation of diseases found (Ridgway, unpublished).

[3] Hibitane—a product of Imperial Chemical Industries Ltd., Alderley Park, Cheshire, England.

Condition	Age	Species
1. Lung worm infestation	Under 1 year	sea lion
		elephant seal
	subadult	harbour seal
2. Malnutrition, gingivitis and parasites	Under 1 year	harbour seal
		elephant seal
3. Gunshot or puncture wounds	All ages	sea lion
4. Perforated gastric ulcers and peritonitis	Adult (Old)	sea lion

Some animals are affected by two or more of these conditions. Animals with severe lung worm infestation and those with perforated gastric ulcers nearly always died. Some of those with gunshot wounds have been saved through surgery and intensive antibiotic medication.

Lung worm infestation in sea lions

The great majority of sick pinnipeds that come to the mainland coast of California from their natural island habitats are young California sea lions. Although a number of attempts have been made to save such animals from death, all have failed.

By the time the animal is sick enough to come to the beach the lung worm infestation is so advanced that little viable lung tissue remains, and pulmonary oxygenation of blood is severely hampered. Fecal examination has revealed numerous larvae. Although these animals often appear to be hungry, they will take food and usually vomit immediately. The lung worm larvae may have caused severe irritation of the upper gastrointestinal tract. Dailey (1970) has described the life cycle of this lung worm *Parafilaroides decorus*, and Simpson and Gardner (1972) have recently discussed the histopathology of a number of cases.

Malnutrition, gingivitis and parasites

Young elephant seals and harbour seals (less than 6 months) occasionally are found on the beach in a very emaciated condition. Malnutrition is probably a principal factor. Geraci (1970) demonstrated the presence of severe thiamine (Vitamin B_1) deficiency in one such group of harbour seals on Sable Island, Nova Scotia. The condition was due likely to the pups having been abandoned or weaned too soon, or to antithiamine compounds in their fish diet.

Whatever the initial cause of malnutrition, such animals eventually are shown to have severe gingivitis with abscess formation in the gums and pus formation around the teeth. Sometimes the eyes are also affected and there is severe purulent conjunctivitis. Culture of the mouth and eye lesions have revealed mixed growths of *Streptococcus*, *Pseudomonas*, and *Proteus* organisms. It is doubtful that these organisms are primary pathogens. More likely, they behave opportunistically and establish a foothold once the seal is debilitated. Many of these starving animals can often be saved with a daily regimen of antibiotic therapy, and by force feeding with a stomach tube using blended fish and vitamins in saline. The seals usually start to eat whole fish within about a week, at which time they may be wormed with thiabendazole and piperazine. Many of the animals that have died have had large numbers of gastrointestinal parasites as well as lung worms.

Gunshot or puncture wounds

Occasionally sea lions are found with circumscribed puncture wounds penetrating the flipper and pectoral area. On one occasion lesions were severely infected and the animals which were examined seemed to be suffering from a generalized septicemia. The white blood cell count was greatly elevated compared to the usual findings (Ridgway, 1972). Antibiotic medication was started immediately and the animals were anesthetized by the method of Ridgway and Simpson (1969). The lesions were thoroughly cleaned and antiseptic applied. Three such animals were saved. One sea lion, in which the radius was broken and the thorax contained lead shot, was killed.

California sea lions are sometimes seen resting on the sea surface with one or more flippers extended in the air. The location and appearance of the wounds we encountered suggest that the sea lions may have been shot while resting in this manner.

Gastrointestinal disorders

Signs of gastrointestinal disturbances, i.e. loss of appetite, vomiting and diarrhea, are a common and bothersome occurrence in pinniped colonies. It is often difficult to establish a diagnosis as the conditions may be primary, or simply a clinical manifestation of some totally unrelated disease.

Certainly foreign bodies, parasites and bacterial infection are high ranking causes, and if accurately diagnosed, may be controlled by anthelminthics, antibiotics, and by surgical intervention.

Untreated or non-responsive diarrhea and vomiting result in dehydration, acid-base imbalance, and possibly salt-wasting. They may also intensify the inflammatory reaction and lead to hemorrhage, erosions, ulcers, and fatal perforations, all of which have been reported or observed.

The following case histories, which reflect some of the techniques used in diagnosing and treating gastrointestinal disorders, serve as repesentative examples of what might be expected to occur in wild and captive pinnipeds.

Dental disease

Stirling (1969) has commented on tooth wear as a mortality factor in the Weddell seal in the Antarctic. It is no doubt important in other species as well. In captivity walruses may grind their teeth on concrete. The enamel can be worn away exposing the pulp cavity to infection. The ice-sawing behavior of the Weddell seal can also cause such problems in captivity.

Gastrointestinal foreign bodies

The literature relevant to stone swallowing and the gastrointestinal foreign-body problem in general has been reviewed by Ridgway (1972). Although foreign-body swallowing cannot be regarded as normal behavior, it has not yet proven to result from parasitism, nutritional disorders or other disease. It does seem likely that at least some of this behavior has its origin in a pathological condition.

When captive animals swallow foreign objects it is important that steps to alleviate the situation not be taken too hastily unless the object is toxic, sharp or in some way could be definitely and immediately injurious to the animal. A rubber ball, for example, might do no harm in a seal's stomach. In time it could disintegrate causing possible problems in the lower intestine. Where warranted, objects can be removed by surgery.

Gastric ulcers

A young California sea lion went off its feed. It spent most of the time resting on its side with its front and rear flippers folded along the body, or resting on its chest with the flippers stretched out to the side. The white blood cell count was within normal range but the hemoglobin concentration was low (13 gm/100 ml). Since the sea lion's behavior suggested abdominal pain, the animal was slightly anesthetized with halothane and a fiber-optic gastroscope was passed into the stomach along with a stomach tube. When the stomach was inflated by blowing on the tube several erosions were observed in the stomach wall.

The following medication was initiated. About 1·5 litres of "gruel" were mixed in a large blender. It contained eight Maalox tablets (magnesium aluminium hydroxide), 500 mg tetracycline, 100 mg thiamine, 50 ml of an essential fatty acid-vitamin supplement (Nutriderm, a product of Norden Laboratories, Lincoln, Nebraska), 500 g herring or mackerel, 500 g of squid, and enough water to make the 1·5 litres. This was given to the animal twice daily by stomach tube. Early in the third week of treatment the animal started taking squid on its own and made an uneventful recovery (Ridgway, unpublished).

Perforated gastric ulcers and peritonitis

Over a period of about seven years, several adult female sea lions have been encountered on the beach at Point Mugu, California. They were slightly to extremely emaciated. The appearance of the teeth and the presence of considerable grey hair about the snout and head suggested that they were quite old. All of these animals died within 24 hours of being taken from the beach.

Rectal temperature after capture was 39·5–41·5°C and the white blood cell count was greatly elevated. Post mortem examination revealed one or more perforated gastric ulcers containing nematodes. The worms from one stomach were identified as *Contracaecum osculatum*. The entire abdomen was filled with purulent fluid and there war a severe peritonitis.

Fiennes (1968) has reported a perforated gastric ulcer in a Baikal seal (*Pusa sibirica*) and Appleby (1962) found a gastric perforation in an elephant seal.

Ulcerative colitis

A California sea lion about three years old was undergoing preliminary training for some diving experiments. For about four months it had been fed wholly on an experimental synthetic ration. The sea lion appeared to like the ration and accepted it as reward for retrieving objects, pressing a paddle, etc. The trainer described the animal's overall behavior as highly excitable.

The animal suddenly went off its food and refused to train. On the following day abdominal pain was evident. The seal would rest on its side with flippers folded along the body, or on its stomach with the flippers extended out to the side. There was considerable trembling and grunting. It frequently changed from one position to another but would not walk or move around normally.

Radiographs revealed that there was no foreign body in the gastrointestinal tract but there did appear to be excess fluid in the abdomen. The animal was put on antibiotic medication but died the following morning.

Post mortem examination revealed perforated ulcers of the colon and severe peritonitis. It is not known whether the animal's temperament or the synthetic ration contributed to its death.

A syndrome of hemorrahagic gastroenteritis

Four California sea lions and one northern fur seal, *Callorhinus ursinus*, began to undergo episodes of severe diarrhea, intermittent vomiting and some respiratory distress after 3 years in captivity on the New England coast. The results of repeated fecal bacterial and parasite examinations were negative, there was no

history of any change in diet, the condition was not responsive to symptomatic treatment. The use of a low level tranquillizer in order to obtain blood proved to be fatal to one of the sea lions. Necropsy revealed interstitial pneumonia, hemorrhagic gastritis, enteritis and colitis, and nephritis. All organs examined showed the presence of microfilaria. Subsequent blood samples from the other animals in the colony revealed marked eosinophilia with or without leucocytosis, and microfilaria which are now highly suspect as being the primary cause of the condition. Treatment with a filaricide is now underway, as is parasite identification; preliminary results are encouraging (Geraci and Wilson, unpublished 1972).

Pancreatic inflammation

In both cetaceans and pinnipeds in captivity there have been occasional deaths that appeared to result from severe inflammation of the pancreas. The large pancreatic duct may allow easy access to some infections that might first become established in the gastrointestinal tract.

Ridgway (1972) has recently discussed the glucose tolerance of several marine mammals and some aspects of the carbohydrate metabolism in relation to the fish diet. There is much to be learned about the pancreatic physiology and an explanation for deaths due to severe pancreatic inflammation is yet to be found.

Liver disease

Geraci (1970) showed that harp seals, *Pagophilus groenlandicus*, in captivity commonly exhibit change in circulatory hepatic cell enzymes which clearly indicates the presence of liver damage. Though such change nearly always accompanies the moult, it also occurs sporadically, sometimes in apparent response to stress. It sometimes occurs as part of a syndrome which resembles infectious canine hepatitis (ICH). Attempts to discover the cause, including ICH serum titres, pesticide analyses, and histopathology, have not been successful. It is likely, though unproven, that a hepatitis virus which is not cross reactive with ICH is responsible for at least some of these clinical problems.

Reproductive disorders

Abortion, ovarian tumors, intrauterine loss of blastocysts (Rand, 1955), prolapsed uterus and postpartum uterine infections are among the reproductive disorders that have been observed. In some species there is an apparent high offspring mortality both *in utero* and during the first year of life.

In California, a recent epidemic of abortions was thought to be associated with leptospirosis. Pyometra has been observed in the California sea lion and corrected by surgical removal of the uterus and ovaries. De Long et al. (1973) have connected abortion and organochlorine pollutants.

Arterial disease

Most marine mammals have a fairly high blood cholesterol level (Ridgway, 1972), yet their marine food supply is relatively low in saturated fats thought to be associated with high cholesterol content and some arterial disease. Prathap et al. (1966) were unable to find any correlation between cholesterol and arterial lesions in the Weddell seal. They found arteriosclerotic lesions in some seals and reported a relatively high cholesterol level in all seals.

Kelly and Jensen (1960) reported on a case of chronic aortis in a California sea lion. Finlayson (1965) discusses a number of cases of arterial disease and reviews some of the earlier literature on the subject. Stout (1969) also discusses ateriosclerosis in pinnipeds.

Scott (1928) reported on the death of a walrus from intrathoracic hemorrhage. Orcutt (1947) mentioned the death of two elephant seals from ruptured blood vessels. Johnston and Ridgway (1969) discussed the probable relationship of parasitism to thrombosis of pulmonary arteries and veins in an elephant seal. We are aware of other cases of elephant seal deaths in which the major post-mortem finding was a ruptured thoracic blood vessel.

Fiennes (1966) reported on a young grey seal, *Halichoerus grypus*, that died of bronchopneumonia. There was a thrombotic occlusion of the left iliac artery. This resulted in the animal being immobile, and pneumonia subsequently developed. "Histological studies suggested that calcification of the arterial wall might have been caused by migrating larvae."

Many of the vascular lesions appear to have resulted from parasitism and this must be considered the primary cause of vascular disease in marine mammals based on information currently available.

Central nervous system

Sodium wasting

A female gray seal about one year of age was operated on under thiopental-halothane anesthesia for the placement of a 3 channel transmitter to telemeter the electroencephalogram (EEG) and brain temperature. Large venous sinuses course through the bone of the dorsal cranium, one of which was accidentally penetrated, resulting in profuse hemorrhage presumably above and below the cranial bones. The cortex was also penetrated with a bone drill. The operation was limited to the left side of the skull.

On the day following the operation, the seal would

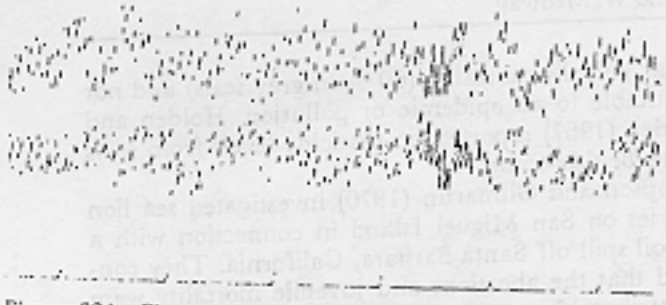

Figure 224. Electroencephalogram of a female seal about one minute after a seizure episode. Time scale in seconds (25 mm/sec). Upper tracing lead 1 and lower tracing lead 2.

hardly move and its head was grossly swollen. The animal drank fresh water greedily from a hose tap supplying water to its pen. The seal remained on her right side and if shifted would immediately turn back to the right side. Chloromycetin (1 g) was given in the muscle, morning and evening, to combat infection.

On the second day following surgery the animal's condition had slightly improved. A blood sample was collected from the intervertebral extradural vein. In order to relieve pressure on the thorax the seal was placed in a small tank in shallow water. During the next few days the seal appeared to improve, but occasionally it had fits ranging from mild trembling to severe tremors or convulsions involving the whole body. EEG records were not obtained during any of these convulsive episodes, but were recorded within a minute after the seizures had occurred. Such records were characterized by high voltage spike and dome activity not unlike that seen in epilepsy (Fig. 224). The records on both channels of the EEG were almost identical, indicating that the seizures probably resulted from a generalized condition rather than a focus as in epilepsy.

Blood analysis showed a marked reduction in plasma sodium and a slight reduction in plasma potassium as compared to presurgical values. It was suspected that the seal might be suffering from cerebral wasting as described by Cort (1965).

The seal was started on twice daily oral injections (by stomach tube) af Zawb-O-Lyte[1], a product used in electrolyte replacement therapy for calf scours. It contains sodium chloride, potassium chloride and magnesium chloride. By the third day after this therapy was instituted, the seizures had ceased and the seal could consume fish on its own. No further signs of seizure-like activity were seen on the EEG during subsequent 3 months when recordings were made from this seal.

[1] Zawb-O-Lyte is a product of Willows Francis Ltd., Lancashire, England.

Cerebral sodium wasting has been described by Cort (1965) from cases of severe cranial injury in humans. Although a more controlled study, including determination of urinary electrolyte excretion, would be necessary to establish that the seal's seizures and sodium depletion were the result of the same condition described by Cort, the treatment undertaken was successful (Ridgway, unpublished).

Geraci (1972b) has described convulsions, seizure-like activity, and sodium depletion in severe thiamine deficiency in harp seals. However, the present seal and 5 others were receiving 50 to 100 mg daily of thiamine during the 10 months of their captivity and all appeared to be normal. In a follow-up report, Geraci (1972 a) modified his original thoughts on the cause of electrolyte imbalance by demonstrating that hyponatremia could be included not only by the stress of thiamine deficiency, but also by a host of entirely unrelated factors. In effect, any physiological or pathological stress is likely to induce hyponatremia, perhaps fatal, in harp seals and in some sea lions maintained in fresh water. Again, the condition can be completely cured by simple oral or parenteral administration of sodium chloride, and it can be prevented by supplementing the diet with the same salt.

It appears that the pinniped central nervous system is quite sensitive to electrolyte imbalance. Until more information on electrolyte metabolism is available it would seem most prudent to maintain pinnipeds in sea water whenever possible.

Other brain lesions

We have mentioned two conditions that can cause seizures, motor incoordination and other signs of CNS disturbance: i.e. cerebral sodium wasting and stress induced hyponatremia. However other conditions have been found that could have produced such signs.

A female grey seal developed a large brain abscess over the left cerebral hemisphere at the site of some intracranial electrodes that had been placed for physiological recordings. The only sign manifested was blindness of the right eye.

Flores-Barroeta et al. (1961) reported on an erratic parasitosis of the brains of four California sea lions by *Contracaecum osculatus*, but no signs were mentioned.

The relative thinness of the seal skull especially in young individuals makes it highly susceptible to cranial injury. Boyd and Campbell (1971) mentioned a cerebral abscess in a gray seal pup, apparently caused by a bite from another seal. They also reported a death due to cerebral hemorrhage.

Ronald et al. (1970) have reported a harp seal death due to polioencephalomalacia based on histopathology, which showed degeneration of cortical neurons and

edema of the cerebrum and cerebellum. The animal had had a severe case of pneumonia two months prior to death.

Neoplasia

A number of tumors have been found in pinnipeds, mostly in captive animals. Fox (1923) reported an adrenal hypernephroma, an adenocarcinoma and a myosarcoma of the mammary gland. Simpson and Ridgway (unpublished data) surgically removed a fibroma from the neck of a California sea lion. They also found an adenocarcinoma of exocrine glandular tissue in the same species. Griner (personal communication) at the San Diego Zoo has found a lymphosarcoma, a granulosa cell tumor, and an adrenocortical carcinoma. Nakajima and Fujimoto (1971) reported on a retro-peritoneal tumor that occupied most of the abdominal cavity of a California sea lion. The tumor weighed 7·3 kg and the histology was described as being compatible with a designation of malignant mesenchymoma. Griner (1971) found malignant leukemic lymphoma in two harbour seals.

Mawdesley-Thomas and Bonner (1970) reported several uterine tumors from a grey seal estimated to be at least 44 years of age. The cervix contained tumors described as uterine leiomyomas an there was a squamous-cell carcimona in a uterine horn. Mawdesley-Thomas (1971) found a 340 g mass replacing the left ovary in a southern elephant seal. It was described as a malignant granulosa-cell tumor. Medway and Sweeney (unpublished data) found a bladder carcinoma in an adult female sea lion.

Most neoplastic conditions appear as incidental findings in captive seals. They are difficult to diagnose, they do not appear with any degree of regularity, and they are either innocuous or incurable without surgery. Therefore, they are generally considered to be of academic rather than practical interest in any pinniped colony.

Pollution

The development of sophisticated and highly sensitive techniques for detecting various compounds in animal tissues has led to the discovery of a number of contaminants throughout the ocean food web. Since pinnipeds occupy high positions in the food web, the assessment of compounds such as DDT metabolites, other organochlorines and mercury have been of interest. We are not aware of any published reports of toxic disease in seals or sea lions resulting from these pollutants, or from oil pollution.

Bonner (1970) investigated seal deaths in Cornwall, England, in 1969. Surveys were made for various contaminants such as those mentioned above. It was concluded that the deaths observed were part of the normal juvenile mortality (60 % in grey seals) and not attributable to an epidemic or pollution. Holden and Marsden (1967) reported on pesticide levels from seals off the British coast.

Simpson and Gilmartin (1970) investigated sea lion rookeries on San Miguel Island in connection with a large oil spill off Santa Barbara, California. They concluded that the abortions and juvenile mortality were not attributable to oil pollution. Others have, more recently, attributed sea lion deaths and abortions to leptospirosis.

Le Boeuf and Bonnel (1971) have found high concentrations of DDT residues in California sea lions. They doubt that DDT contamination resulted in sea lion deaths since similar concentrations were found in living animals.

DDT residues, organochlorines, and mercury can have direct toxic effects on the CNS. Oil, if ingested, could possibly cause abortion and mortality of the newborn. DeLong et al. (1973) have found organochlorine pesticides and polychlorinated biphenyl residues 2 to 8 times higher in premature parturient females and pups than in tissues of females and full term pups on San Miguel Island, California in 1970. Thus there appears to be a relationship between premature pupping and pesticide residues. It would be helpful if we knew more about the toxicology of these agents in pinnipeds.

As an ancillary part of a nutritional study in pinnipeds, Geraci (1970) found a direct relationship between DDT concentrations in food fish and the deposition of the pesticide within various tissues in harp seals. A surprising aspect of the investigation was that within very brief periods (2–6 months) during which the seals were on very low pesticide-intake diets, they appeared to metabolize and/or eliminate most of their tissue residues. The sample size was small, and the results therefore inconclusive; it would appear however, that the high concentrations of DDT which have been reported in pinnipeds may be a reflection of current and continuous ingestion of the substance.

Microbial diseases

Viruses

The term "viral disease" is often misused to identify conditions of unknown origin. Some of these conditions are undoubtedly due to viruses, but as such they are difficult to diagnose and probably become masked by secondary bacterial and parasitic invasion.

To our knowledge, the only pinniped disease whose viral identity has been confirmed, is sealpox, a naturally occurring disease which has been reported in wild and captive sea lions, harbour seals and South American sea lions, *Otaria byronia*, (Wilson, Dykes,

and Tsai, 1972). The disease is characterized by proliferative skin lesions, intracytoplasmic inclusion bodies in stratum spinosum cells, and dumbbell- shaped pox virus virions within the inclusions. Clinically the condition is manifested by skin nodules. Uncomplicated cases are not known to be fatal, and there is no known treatment except intensive nursing and elimination of any concurrent disease that may cause debilitation of the host.

Rickettsia and pleuropneumonia-like organisms (PPLO)

To our knowledge, there have been no confirmed reports of pinniped diseases caused by these organisms. Ridgway (1972) lists one reference to salmon disease which was discussed by Farrell in 1970.

Bacterial and mycotic disease

It seems best to begin this discourse on bacterial diseases by indicating its role in pneumonia, a common killer of most marine mammals in captivity. Despite its frequency of occurrence, there have been no studies on the exact course of events leading to fulminating clinical pneumonia in pinnipeds. In most cases it is probably not a primary condition, but may be a secondary manifestation of some totally unrelated debilitating disease, or the result of extensive lung or respiratory tract damage from mechanical manipulation (e.g. endotracheal intubation) or destructive parasitic invasion.

Dailey (1970) has reviewed the literature on respiratory tract parasites in pinnipeds. In a study of 74 seals and sea lions, Doetschman (1940) showed an incidence of approximately 70 % lung and nasal passage infections due to 3 species of mites. Seawright (1964) relates the cause of death in a Tasmanian fur seal *Arctocephalus pusillus doriferus,* to pneumonia and asphyxiation from excessive mucus secretion in response to *Orthohalarachne attenuata,* a pulmonary tract mite. Lung nematode infestation such as is caused by *Parafilaroides decorus* (Morales and Helmboldt 1971) is a more common cause of verminous pneumonia.

Sea lions often succumb to the direct insult of this parasite. Bacteria frequently assume a secondary but dominant role in infection. It would be unreasonable to list them by name, as bacteria of all descriptions have been implicated in pinniped pneumonia. Some of the more notorious are *Mycobacterium tuberculosis, Salmonella typhimurium, Staphylococcus* sp, *Streptococcus* sp, and *Pseudomonas* sp.

The diagnosis of pneumonia can be made without too much difficulty if it is sufficiently advanced. Animals evidence signs of respiratory distress, nasal discharge, and perhaps coughing episodes. Unfortunately by the time the disease becomes clinically obvious, it may have progressed to an irreversible stage. Nevertheless treatment should be directed toward eliminating the specific organism.

Bacterial and mycotic infections are certainly not restricted to pinniped lungs. *Staphylococcus* has been isolated from chronic alveolitis in a walrus (Bartsch and Frueh, 1971) and from skin granulomas in a harp seal (Wilson and Long, 1970). Salmonellosis has been implicated in bacillary dysentery in fur seals (Jellison and Milner, 1958) and in septicemia in a hooded seal (Geraci, unpublished).

The presence of these organisms suggests the possibility that pinnipeds may be a source of communicable disease. This consideration certainly warrants more attention than has been accorded in the past. Recent studies have revealed the presence of leptospirosis among California sea lions (Vedros et al., 1971; Northway, 1972), North American blastomycocis in a northern sea lion (Williamson, Lombard, and Getty, 1959) and actinomycocis in a harbour seal (Schmitt, 1962). One must consider these diseases as being potentially dangeous not only to other animals in the colony, but also to attendants and others involved with animal handling. A final and incongruous note to this suggestion is the finding by Kelly and Jensen (1960), Prathap et al. (1966), and Stout (1969) from histopathological and serological evidence that some arterial lesions found in pinnipeds are similar to those of tertiary syphilis in man.

Metabolic disorders

Ridgway (1972) has presented a review of marine mammal metabolism. Their peculiar physiological demands make it clear that any degree of environmental tampering, such as changing salinity, reducing the quality of food, or imposing unrealistic stress can induce injurious metabolic disorders. To date, very few of these possible hazards have been assessed adequately. Thiamine deficiency induced by feeding thiaminase-containing fishes has been morphologically and biochemically confirmed only recently (Rigdon and Drager, 1955; Geraci, 1972a), but there is no firm evidence of any other vitamin deficiency. Dehydration and electrolyte imbalance have been described in otariid and phocid seals, along with some ideas on possible mechanisms. (Ridgway, this volume; Hubbard, 1968; Geraci, 1972b). As yet, the true causes have not been confirmed. "Stress-induced" hypoglycemia is known to occur in pinnipeds (Brown, 1962; Hubbard, 1968), as are renal calculi in ringed and Weddell seals (Geraci, unpublished), but the cause and pathogenesis are unknown. Deficiencies of calcium and vitamins A, D and C and dietary histamine

toxicity are thought to occur, but they too remain open questions.

Metabolic disorders are the least understood and least investigated problems in pinnipeds, an understandable situation in view of the high cost of current research in this area. Nevertheless it is safe to assume that most of the conditions we deal with in pinniped colonies have a metabolic basis either as a primary or secondary event in the course of disease. It would therefore be beneficial to understand this aspect in more detail.

DISEASE DIAGNOSIS, PREVENTION AND TREATMENT

One of the more challenging experiences for a veterinarian is to attempt to diagnose marine mammal illnesses. Generally one must rely on quite subtle physical changes and on variations in behavioral characteristics which are usually quite predictable. Unfortunately, as is often the case, by the time the animal begins to manifest overt signs of illness, the disease has progressed to an irreversible stage.

This type of response, which characterizes not only pinnipeds, but a wide variety of wildlife species, has been termed "staying power". It undoubtedly has great selective advantage in natural surroundings, and at least serves to prevent advertisement to a predator, of weakness within the group. In captive surroundings, however, it offers a distinct disadvantage. Frequently, a seal may die of fulminating septicemia and purulent pneumonia within two hours of a normally ingested meal, having shown no previous signs of illness. This situation has forced many investigators to rely upon routine blood samplings for the purpose of evaluating subclinical conditions. Using this technique, a laboratory diagnosis often precedes by 1–3 weeks a clinical "suspicion". Data on normal values for most "laboratory" marine mammals is available. Hematologic response to bacterial infections is much the same as in other mammals, with increased white cell counts, cell shifts, etc. Anemias are a common finding in certain seal colonies; although the cause cannot always be determined, an early diagnosis followed by symptomatic treatment is often successful. Some of the plasma chemical constituents serve as very reliable indicators of health. The blood changes in clinical hyponatremia have, at times preceded clinical signs by as much as one month.

Circulating enzyme levels are accurate determinants of organ integrity; for example, liver and muscle destruction is accompanied by elevations in plasma enzymes which are released by the corresponding tissue cells, as in terrestrial mammals. Renal function can be easily assessed by determining creatinine and urea nitrogen levels. In short, blood constituents can be expected to respond in a manner similar to their counterparts in other laboratory mammals. There are a few and notable exceptions; for example uric acid is unpredictably high in some seals, glucose levels are high in both Cetacea and pinnipeds, as are concentrations of lactic dehydrogenase enzymes. All of these "sophisticated" diagnostic tests are intended not to supplant, but to complement the reliable and age-tested methods of thorough case-history study and physical examination.

Once a diagnosis has been established, whether definitive or otherwise, basic therapeutic measures can be instituted without any real consideration for species. Antibiotic therapy should follow the indications from culture and antibiotic sensitivity studies. There is no antibiotic, antibacterial, chemotherapeutic, or biological agent of which we are aware that is peculiarly contraindicated in marine mammals, transquilizers and anesthetic agents excepted. Dosage is often based more on lean body weight, which often constitutes 45 % to 70 % of the total. The higher basal metabolic rate of some species must be considered. In addition the large total blood volume may be a factor in maintaining useful blood levels of antibiotics. The increased glomerular filtration and diuresis that occur after a fish meal can cause more rapid excretion of some antibiotics. Thus total dosage and frequency must be increased. Fluids may be administered with ease by stomach tube or intraperitoneal infusion. Skin conditions are, at best, difficult to treat and it may be more feasible to medicate the water than to remove the animal periodically. In all cases, one should choose a therapeutic program which involves the least amount of animal handling or contact.

There is virtually no limit to the quality of the medical program one wishes to institute. When necessary, pinnipeds can be maintained out of water for indefinite periods, providing they can be kept clean, cool and occasionally showered. As a holding facility, a modified dog kennel can serve the purpose easily.

Surgical procedures of all descriptions have been carried out successfully on pinnipeds. Owing to their peculiar respiratory physiology, the only real precaution should be directed toward providing positive pressure inhalation. Volatile anesthestic agents are superior, whereas barbiturates and injectable agents in general are contraindicated, unless used by experienced individuals under controlled laboratory conditions.

REFERENCES

Appleby, E. C. 1962. A case of gastric perforation by a foreign body in an elephant seal (*Mirounga leonina*). Nord. Vet. Med., 14, Suppl. 1:164–65.

Barrisch, R. C. & Frueh, R. J. 1971. Alveolitis and pulpitis of a canine tooth in a walrus. Am. Vet. Med. Assoc., J., 159: 575–77.

Bonner, W. N. 1970. Seal deaths in Cornwall, autumn 1969. Nat. Environ. Res. Counc., (NERC). Publ., Ser. C, 1.
Boyd, J. M. & Campbell, R. N. 1971. The grey seal (*Halichoerus grypus*) at North Rona, 1959 to 1968. J. Zool., 164:469–512.
Brown, D. H. 1962. The health problems of walrus calves and remarks on their general progress in captivity. Int. Zoo Yearb., 4:13–23.
Cort, J. H. 1965. Electrolytes fluid dynamics and the nervous system. Academic Press, New York. 230 pp.
Dailey, M. D. 1970. The transmission of *Parafilaroides decorus* (Nematoda: Metastrongyloidea) in the California sea lion (*Zalophus californianus*). Helminthol. Soc. Wash., Proc., 37: 215–22.
DeLong, R. L., Gilmartin, W. G. & Simpson, J. G. 1973. Premature births in California sea lions associated with high organochlorine pollutant residue levels. Science, New York, 181:1168–69.
Doetschman, W. H. 1940. The occurrence of mites in pinnipeds, including a new species from the California sea lion, *Zalophus californianus*. J. Parasitol., 26:23.
Eriksen, E. 1962. Diseases of seals in the Copenhagen Zoo. Nord. Veterinarmed., 14, Suppl. 1:141–49.
Fiennes, R. W. 1966. Report of the Society's pathologist for the year 1964. J. Zool. Lond., 148:363–80.
Fiennes, R. W. 1968. Report of the Society's pathologist for the year 1965. J. Zool. Lond., 154:173–91.
Finlayson, R. 1965. Spontaneous arterial disease in exotic animals. J. Zool., 147:239–43.
Flores-Barroeta, L. Hidalgo-Escalante, E. & Oleae, R. 1961. Nematodes from birds and mammals. 4(1) Erratic parasitosis in *Zalophus californianus* from Asuncion Island, Baja California, Mexico. Helminthologia, 3:112–16.
Fox, H. 1923. Diseases in captive wild mammals and birds. Lippincott, Philadelphia.
Gallacher, J. B. & Waters, W. E. 1964. Pneumonia in grey seal pups at St. Kilda. Zool. Soc. Lond., Proc., 142:177–80.
Geraci, J. R. 1970. The effects of fish induced thiamine deficiency on the harp seal, *Pagophilus groenlandicus*. McGill Univ., Ph.D. Thesis.
Geraci, J. R. 1972a. Experimental thiamine deficiency in captive harp seals, *Phoca groenlandica* induced by eating herring, *Clupea harengus* and smelts, *Osmerus mordax*. Can. J. Zool., 50:179–95.
Geraci, J. R. 1972b. Hyponatremia and the need for dietary salt supplementation in captive pinnipeds. Am. Vet. Med. Assoc., J., 161:618–23.
Griner, L. A. 1971. Malignant leukemic lymphoma in two harbor seals (*Phoca vitulina geronimensis*). Am. J. Vet. Res., 32:827–30.
Holden, A. V. & Marsden, K. 1967. Organochlorine pesticides in seals and porpoises. Nature, Lond., 216:1274–76.
Hubbard, R. C. 1968. Husbandry and laboratory care of pinnipeds. pp. 299–338. *In* The behaviour and physiology of pinnipeds. Ed. by R. J. Harrison, R. C. Hubbard, R. S. Peterson, C. E. Rice and R. J. Schusterman. Appleton-Century-Crofts, New York.
Hubbard, R. C. & Poulter, T. C. 1968. Seals and sea lions as models for studies in comparative biology. Lab. Anim. Care, 18:288–97.
Jellison, W. L. & Milner, K. C. 1958. Salmonellosis (bacillary dysentery) of fur seals. J. Wildl. Manage, 22:199–200.
Johnston, D. G. & Ridgway, S. H. 1969. Parasitism in some marine mammals. Am. Vet. Med. Assoc., J., 155:1064–72.
Kelly, A. L. & Jensen, D. 1960. Chronic nortitis in the California sea lion, *Zalophus californianus*. Nature, Lond., 186:731.
Larsen, S. 1962. A survey of post-mortem findings in pinnipedia, autopsied during the 10-year period 1952–1961. Nord. Vet Med., 14, Suppl. 1:150–60.
Le Boeuf, B. J. & Bonnell, M. L. 1971. DDT in California sea lions. Nature, Lond., 234:108–10.
Mawdesley-Thomas, L. E. 1971. An ovarian tumor in a southern elephant seal (*Mirounga leonina*). Vet. Pathol., 8:9–15.
Mawdesley-Thomas, L. E. & Bonner, W. N. 1970. Uterine tumors in a grey seal (*Halichoerus grypus*). J. Pathol., 103:205–08.
Morales-Agacino, E. & Heimbolct, C. F. 1971. Verminous pneumonia in the California sea lion (*Zalophus californianus*). J. Wildl. Dis., 7:22–27.
Nakajima, M. & Fujimoto, Y. 1971. A case of retro-peritoneal tumor of a California sea lion (*Zalophus californianus*), compatible with malignant mesenchymoma. Enoshima Marineland Reports, 13:1.
Northway, R. B. 1972. Leptospirosis in a California sea lion. Vet. Med. Small Anim. Clin., 67:138–41.
Orcutt, E. 1947. Why autopsy? Zoonooz, 20(5):6.
Prathap, K., Ardlie, N. G., Peterson, J. C. & Swarz, C. J. 1966. Spontaneous arterial lesions in the Antarctic seals. Arch. Pathol., 82:287–96.
Rand, R. W. 1955. Reproduction in the female cape fur seal, *Arctocephalus pusillus* (Schreber). Zool. Soc. Lond., Proc., 124, Part 4:717–40.
Ridgway, S. H. 1972. Homeostasis in the aquatic environment. pp. 590–747. *In* Mammals of the sea: biology and medicine. Ed. by S. H. Ridgway. Charles C. Thomas, Springfield, Ill.
Ridgway, S. H. & Simpson, J. G. 1969. Anesthesia and restraint for the California sea lion *Zalophus californianus*. Am. Vet. Med. Assoc., J., 155:1061–63.
Rigdon, R. H. & Draper, G. A. 1955. Thiamine deficiency in sea lions (*Otaria californiana*) fed only frozen fish. Am. Vet. Med. Assoc., J., 127:473–75.
Ronald, K., Johnson, L., Foster, M. E. & Vander Pol, D. 1970. The harp seal, *Pagophilus groenlandicus* (Erxleben, 1777). I. Method of handling, medicine and diseases in captivity. Can. J. Zool., 48:1035–40.
Schmitt, J. 1962. Aktinomykose bei einer Mähnenrobbe. Nord. Vet Med., 14, Suppl. 1:170–73.
Scott, H. H. 1928. Report on the deaths occurring in the Society's gardens during the year 1925. Zool. Soc. Lond., Proc., 1928:81.
Seawright, A. A. 1964. Pulmonary acariasis in a Tasmanian fur seal. J. Comp. Pathol. Therap., 74:97–100.
Simpson, J. G. & Gilmartin, W. G. 1970. An investigation of elephant seal and sea lion mortality on San Miguel Island. Bioscience, 20:389.
Simpson, J. G. & Gardner, M. B. 1972. Comparative microscopic anatomy of selected marine mammals. pp. 298–418. *In* Mammals of the sea: biology and medicine. Ed. by S. H. Ridgway. Charles C. Thomas, Springfield, Ill.
Stirling, I. 1969. Tooth wear as a mortality factor in the Weddell seal, *Leptonychotes weddelli*. J. Mammal., 50:559–65.
Stout, C. 1969. Arteriosclerosis in exotic Carnivora and Pinnipedia. Am. J. Pathol., 57:673–87.
Vedros, N. A., Smith, A. W., Schonewald, J., Migaki, G. & Hubbard, R. C. 1971. Leptospirosis epizootic among California sea lions. Science, New York, 172:1250–51.
Williamson, W. H., Lombard, L. S. & Getty, R. E. 1959. North American blastomycosis in a northern sea lion. Am. Vet. Med. Assoc., J., 135:513–15.
Wilson, T. M. & Long, J. R. 1970. The harp seal, *Pagophilus groenlandicus*. (Erxleben, 1777). 12. Staphylococcal granulomas (Botryomycosis) in harp seals. J. Wildl. Dis., 6:155–59.
Wilson, T. M., Dykes, R. W. & Tsai, K. S. 1972. Pox in young, captive harbor seals. Am. Vet. Med. Assoc., J., 161:611–17.